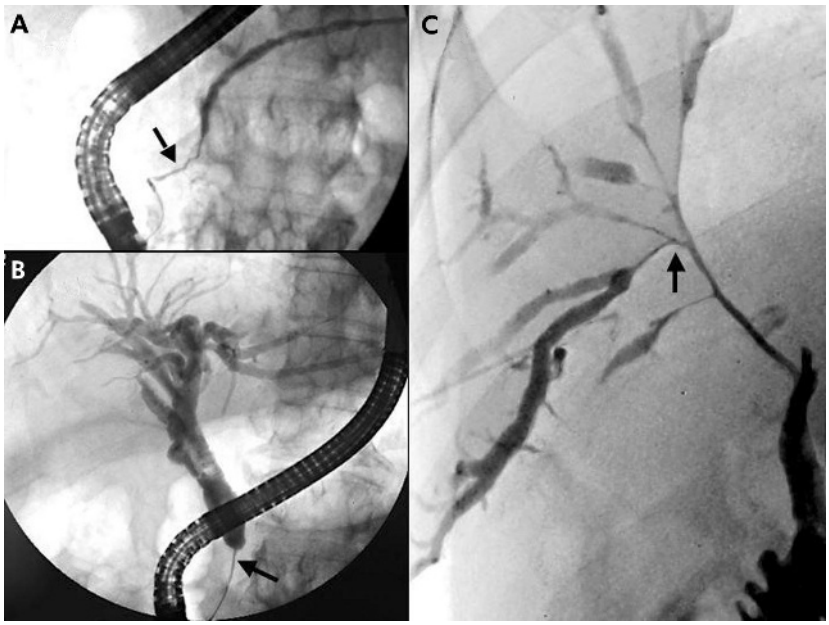

Figure 1 (A, B) Preoperative endoscopic retrograde cholangiopancreatography showing an irregular stricture (arrow) of the pancreatic duct in the pancreatic head (A) and intrapancreatic biliary stenosis (arrow) with prestenotic dilatation of the intra- and extrahepatic bile ducts (B). (C) Postoperative right sided percutaneous cholangiography showing a narrow common bile duct and multiple filiform strictures (arrow) of the bile ducts at the hepatic division with dilatation of the peripheral bile ducts.

continued. The patient remained in good health with normal liver values at regular follow up visits for three years.

In our exceptional case, postsurgical severe sclerosing cholangitis developed after pylorus sparing pancreaticoduodenectomy. In contrast with other similar case reports in which steroids were used,[3] complete remission of sclerosing cholangitis occurred in our patient without steroid therapy. This self limiting postoperative course is remarkable, suggesting that steroid therapy is not always necessary in this situation. Interestingly, immunoglobulin G of the patient's serum reacted both with the pancreas and biliary epithelia,[4] supporting the diagnosis of autoimmune pancreatitis with associated sclerosing cholangitis. The pathogenetic role of this immunohistochemical finding needs to be clarified. In summary, it is important to recognise autoimmune pancreatitis and associated sclerosing cholangitis, which can suddenly occur after pancreatic resection.

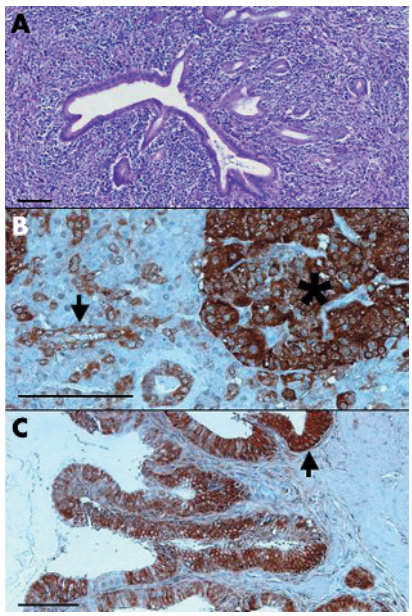

Figure 2 (A) The patient's pancreas, showing a pancreatic duct surrounded by a dense lymphoplasmacytic infiltrate. (B, C) Immunoglobulin G of the patient's serum bound to normal islets of Langerhans (*), and normal epithelia of the pancreatic ducts (arrow) and gall bladder (arrow in (C)). Bar 100 μm.

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Disappointing results of combination therapy for HCV?

Hepatitis C virus (HCV) infection is a common cause of liver disease in the UK. HCV can cause liver failure and liver cancer, and is a frequent indication for liver transplantation. HCV infection can be cured by antiviral therapy. Standard therapy includes the combination of pegylated interferon and ribavirin (PEG-IFN/RIBA) for 24 or 48 weeks, according to HCV genotype.[1–3] A sustained virological response (SVR), defined as undetectable serum HCV RNA six months after cessation of therapy, indicates successful treatment, almost certainly cure. Published registration trials reported encouraging SVR rates of 42–52% for genotype 1 and 77–88% for genotypes 2/3.[1–3]

However, analysis of the combined experience of six European and US centres reported significantly worse results.[4] In that report, the response rate to treatment of genotype 1 infection with pegylated IFN alpha 2a and ribavirin was only 36%. Also, a large US based prospective randomised study reported response rates of 29–34%, depending on the dose of ribavirin used.[5] In contrast, a Canadian study that prospectively tracked patients receiving HCV treatment reported SVR rates of 47.1% for patients with genotype 1 infection.[6] Pretreatment counselling requires discussion with the patient of the likely response to treatment. Most physicians probably quote to their patients the success rates from the registration studies. It is important to be able to discuss the chance of cure as it stands before embarking on treatment, and also the chance of cure if the patient is able to tolerate and complete the proposed course of treatment.

We undertook a casenote and database review of consecutive patients at our centre treated with PEG-IFN/RIBA therapy between 2000 and 2005. We report an intention to treat (ITT) analysis of the results of treatment for this cohort. We also examined baseline clinical and laboratory characteristics of our treated cohort to identify those patients who would have been eligible for treatment according to the inclusion and exclusion criteria for one of those studies.[1] In practice, these criteria identify patients with more advanced liver disease, a difficult to treat (DTT) population. The results of treatment for the proportion of our cohort that would have been suitable for treatment in that registration study were examined. Finally, we examined the incidence and reasons for treatment intolerance or non-compliance.

A total of 243 patients were treated and had six months of follow up; 70% of patients were male and median age was 44 years (range 17–68). The genotype distribution was genotype 1–38%, genotype 2–10%, and genotype 3–49%.

Table 1 Outcome of patients in the study

Genotype	SVR (ITT)	SVR rate (%)	Treatment completed (outcome known)	SVR (treatment completed)	SVR rate (%)	SVR excluding DTT (ITT)	SVR rate (%)
G1 (n = 92)	26/92	28	66	24/66	36	21/64	33
G2 (n = 23)	18/23	78	17	15/17	88	11/13	85
G3 (n = 118)	69/118	59	92	66/92	72	51/75	68
Other (n = 10)	9/10	90	8	7/8	88	3/3	100
Total (n = 243)	122/243	50	183	112/183	61	86/155	65

Ten patients were genotype 4, 5, or 6, or of mixed genotypes. Of 190/243 patients for whom fibrosis stage was known before treatment, 64 (26%) had established cirrhosis.

In our study 186/243 (77%) patients completed the proposed treatment duration. Outcome is known for 183 patients (table 1). On ITT analysis, SVR rates of 28% were observed for genotype 1, 78% for genotype 2, 59% for genotype 3, and 90% for other genotypes. For patients who completed the proposed duration of treatment, response rates were 36% for genotype 1, 88% for genotype 2, 72% for genotype 3, and 88% for other genotypes. Of particular interest were the 57 (23%) patients who were unable to complete the duration of treatment that was proposed at baseline. This is a higher rate of attrition than in published clinical trials (for example, 14% in Manns and colleagues[1]). The most frequent reasons for withdrawal were depression or an inability to tolerate side effects. Treatment cessation for neutropenia, anaemia, or thrombocytopenia was infrequent, although many patients required dose reduction during treatment. Inclusion/exclusion criteria adopted by Manns and colleagues[1] identified 88/243 (36%) patients as DTT. When DTT patients were excluded from the analysis, the SVR achieved was superior to the ITT SVR results—33% for genotype 1, 85% for genotype 2, and 68% for genotype 3.

On ITT analysis, the results of HCV treatment at our centre appear significantly worse than those in the published literature. This appears to be true for patients with genotype 1 infection in particular. In part, we believe that inferior results reflect inclusion in our cohort of patients with more DTT disease. These patients have more advanced liver disease, with more imminent serious clinical events, and a more urgent need for viral eradication. Despite predictably inferior response rates, they are worthy recipients of antiviral treatment. However, they are more often intolerant of treatment, need more frequent dose adjustment, and are less likely to complete the course of treatment. Analysis of SVR after exclusion of patients with DTT infection showed improved response rates for all genotypes.

A relatively high proportion of our cohort failed to complete the planned course of treatment. Patients with genotype 2/3 infection were more likely to complete the planned duration of therapy than patients with genotype 1. This probably reflects greater difficulty in tolerating the demands of 12 months of treatment for genotype 1 infection compared with six months for genotypes 2/3. Psychological intolerance, mainly depression, appeared quite frequent. A more intensive pretreatment evaluation of psychological status may help to identify individuals whose ability to cope with treatment would be enhanced by antidepressant therapy and/or psychological support. Finally, it is essential that treatment centres know their own results which should be shared with patients at the time of pretreatment counselling.

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Leukotriene receptor antagonists as potential steroid sparing agents in a patient with serosal eosinophilic gastroenteritis

Eosinophilic gastroenteritis is a heterogeneous and uncommon disorder characterised by eosinophilic inflammation of the gastrointestinal tissues. The location and depth of infiltration determine its various manifestations and later serve as the basis for its classification as mucosal, muscular, and serosal forms of eosinophilic gastroenteritis. The gold standard for diagnosis is an endoscopic biopsy showing prominent tissue eosinophilia. Gastrointestinal mucosal involvement causes malabsorption, protein losing enteropathy, and diarrhoea. Infiltration of the muscular layer of the bowel wall may cause gastric outlet or small bowel obstruction. Serosal involvement causes exudative ascites rich in eosinophils; this is the least common form and is usually diagnosed by laparoscopic examination and biopsy of the whole intestinal wall.[1 2]

We present the case of an 18 year old male student with a history of allergic rhinitis presenting with abdominal pain, nausea, and low grade fever, which started a few weeks before his admission. Medical history was unremarkable for any other serious disease. Laboratory findings showed leucocytosis with a high percentage of eosinophils (81%); skin prick testing showed sensitivity to Dermatophagoides pteronyssinus and ragweed pollen; total immunoglobulin E in serum was three times the normal level (311 kI/l); and a bone marrow aspiration specimen showed increased numbers of mostly mature eosinophils (62%) in the differential cell count. These findings suggested an atopic constitution.

Endoscopic examination of the whole gastrointestinal tract, histological analysis, and abdominal computed tomography showed no pathological features. Sonographic abnormalities were discovered mostly in the right lower quadrant of the abdomen where a small amount of ascites was found together with nodular deposits on the parietal peritoneum in the same region, indicating peritoneal inflammation (fig 1). Cytological examination of ascites revealed numerous eosinophils (differential count 91% eosinophils) and a few mesothelial cells in the highly cellular cytospin preparation. Sonographic visualisation of nodular peritoneal deposits associated with eosinophilic ascites, peripheral blood eosinophilia, and atopic constitution permitted a diagnosis of the serosal form of eosinophilic gastroenteritis.

Two weeks of oral prednisolone 20 mg/day brought relief to the patient's symptoms, with normalisation of laboratory parameters and sonographic findings. Two months later the patient suffered a relapse with the same abdominal symptoms. Again, the same diagnostic procedure was done, and therapy with oral prednisolone was effective. During the next relapse of the serosal form of eosinophilic enteritis, which was accompanied by a relapse of rhinitic symptoms, we introduced leukotriene receptor antagonists (montelukast 10 mg/day) with the aim of reducing corticosteroid therapy. After four weeks of this therapy the patient achieved complete clinical and laboratory remission, which